

HISTORY OF LUNG DISEASES OF COAL MINERS IN GREAT BRITAIN: *PART III, 1920-1952

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All sciences are connected ; they lend each other material aid as parts of one great whole, each doing its own work not for itself alone but for the other parts, as the eye guides the whole body and the foot sustains it and leads it from place to place. As with an eye torn out or a foot cut off, so it is with the different departments of knowledge ; none can attain its proper result separately, since all are parts of one and the same complete wisdom.

ROGER BACON (1214-1294).

By 1920, as previously recounted, simple anthracosis due to the inhalation of coal dust had ceased to be regarded as a serious condition and the whole emphasis, as far as occupational disease of the lungs was concerned, was placed on free silica and silicosis. Referring to this period Fisher (1935) has very aptly spoken of the creeping of "the shadow of silicosis over the coal-mining industry". The shadow was first noticed in the Somerset coal-field where Mr. Fred Swift, miners' agent at Radstock, directed attention to a number of local underground workmen, who were suffering from a severe, distressing lung disease which, in the opinion of the local doctors, was due to their occupation, namely branching. This work involves machine drilling in driving roads through highly siliceous sandstones—Pennant rock—locally known as "greys." In 1924 one of these workmen died and Sir Kenneth Goadby, an acknowledged expert in pulmonary diseases, following examination of the lungs, pronounced the disease to be silicosis.

Following this Fisher (1935) records that :

"In 1925, twelve patients, all of whom had been rock-drillers, were examined by x-ray and a number of men working in the same mines who had done no rock-drilling were chosen as controls and examined in the same manner. All the rock-drillers but one were found definitely to be suffering from silicosis, the one case being doubtful."

In the light of present experience the report on the controls is of considerable interest :

"The pictures show a very different condition to

those suffering from silicosis. In the present series no dense mass of fibrous tissue is seen, though a certain amount of peri-bronchial thickening is observed and the lungs are not well expanded. In my opinion, however, the signs present are frequently seen in routine examinations and consistent with good health and in no way the result of occupation."

Meanwhile certifications by the Silicosis Medical Board and a local study of the problem by Kemp and Wilson (1946) have amply confirmed the serious incidence of silicosis among Somerset miners engaged in drilling and blasting in Pennant rock.

Almost simultaneously Middleton (1920), working as a tuberculosis officer, recorded the toll of silicosis among the corresponding group of men, hard ground workers, in South Wales coal mines. A few years later this was underlined by Tattersall (1926) who wrote :

"I was, with increasing frequency, encountering a type of disease which was either not tuberculous or, when proved to be tuberculous, was of a type quite different in onset, course and x-ray findings from what one may call the ordinary and straightforward case of pulmonary tuberculosis."

From the same area, Lyle Cummins (1927) recorded that :

"Coal miners, as a class, are nowadays much less liable to respiratory disease than other workers exposed to 'dust'; so much so that coal dust in itself may be regarded as harmless or even beneficial in the concentration encountered in up-to-date mines."

He did, however, recall the opinion of Mavrogordato in South Africa :

"Any dust inhaled in sufficient quantity will get ahead of any possible elimination from the lungs. He [Mavrogordato] attributes the escape of the

* Part I, 1800-1875, was published in 1951 (8, 127) ; Part II, 1875-1920, in 1952 (9, 93).

collier, under modern conditions, to the fact that in the course of ventilation the dust concentration is diluted and the dust blown away from where the men are at work."

This induced Cummins to comment :

"Coal dust has earned the reputation of being harmless because of the ease with which it is got rid of from the healthy lung in the concentrations usually encountered in coal mines to-day. Where any factor exists to hinder its elimination, however, coal dust is capable of accumulating in the lungs and leading to serious or even fatal disease."

In their favourable view of existing environmental conditions in the mines these two distinguished experts had overlooked that the deepening and extension of the mines, the working of thinner seams, and the progressive increase of machine mining were all contributing to a serious increase in dustiness, which more than offset the advantages of better ventilation.

Despite the almost complete concentration of doctors on silica dust, the possible deleterious effects of coal dust had not escaped the attention of others. Thus the secretary of the Coal Trimmers' Union invited Collis and Gilchrist (1928) to analyse the 426 deaths which had occurred among Cardiff, Penarth, and Barry coal trimmers between 1910 and 1926. (Coal trimmers are dock workers who fill coal into ships' bunkers and cargo holds.)

Collis and Gilchrist concluded that coal trimmers in South Wales "succumb in excess from bronchitis and pneumonia but not from phthisis". They also recorded that "x-ray examination disclosed that, after years of work, the lungs of coal trimmers are not normal, and exhibit signs similar to those widely regarded as characteristic of silicotic fibrosis".

At this point it is necessary to notice briefly several matters, which had already begun to influence, and, in due course, were almost to dominate the story of the pneumoconioses.

The Development of Associated Influences

On January 23, 1896, William Conrad Roentgen announced his epochal discovery of "a new kind of ray" or as he designated them "x-rays". At first these rays were applied mainly in the diagnosis of fractures and in the detection of radio-opaque foreign bodies such as stones in the bladder. Almost immediately experiments were made to use the method in the investigation of diseases of other structures and organs. Thus in 1907 Summons reported his observations on the value of x-ray examination of the lungs in the investigation of miners' phthisis at Bendigo, West Australia (metalliferous mining). Thereafter the method was increas-

ingly used, but until about 1930 it was rather unreliable except for gross pathological changes. In recent years, however, due to better equipment and accessories together with greater experience in the correlation of morbid anatomical and x-ray changes in the lungs, the method has proved of real value, so much so that no examination of the lungs can now be considered complete unless it includes adequate radiographic investigation.

Almost coincident with the advance of this discovery was the development of gold-mining on the Witwatersrand, which in the course of a few years revealed the deadly dangers of dry drilling in quartz ore-bodies. The South African Miners' Phthisis Medical Bureau was instituted in 1916 and since then its members, in association with the staff of the Medical Research Institute at Johannesburg, have made outstanding contributions on all aspects of pneumoconiosis, particularly silicosis.

In 1919, under the Refractories Industries (Silicosis) Scheme, for the first time in Great Britain dust disease of the lungs, silicosis, was recognized as an industrial disease under the Workmens Compensation Acts. The scheme, as its title denotes, was in respect of silicosis and restricted entirely to certain workmen employed in this particular group of industries. However, it established a precedent which stimulated the trades unions to press for extension of the legislation to other industries. Coal miners were included in the Various Industries (Silicosis) Scheme, 1928, but only those men who, failing agreement with their employers, could prove to the satisfaction of the county court judge or other arbitrator that, during the employment to which the disease was alleged to be due, they had been exposed to the dust of silica rock. The disease was defined as silicosis or silicosis accompanied by tuberculosis and compensation was payable only in respect of total disablement or death by reason of the disease.

Coal Miners' Pneumoconiosis Recurs

Now to resume the story of lung diseases in coal miners. In 1926 Cummins had indicated that under certain circumstances, coal dust, which under normal working conditions appeared to be harmless, was capable of accumulating in the lungs and leading to serious or even fatal disease. Three years later, Cummins and Sladden (1930) published the results of "An Investigation into the Anthracotic Lungs of Coal Miners in South Wales". Reviewing past contributions they recount :

"Coal dusts then came to be accepted, both on statistical and experimental grounds as harmless and possibly beneficial, while anthracosis tended to be

relegated to the limbo of departed pathological problems connected with the faulty mining conditions of the past."

The following pathological description is quoted in full for comparison with those recorded just a century previously (Meiklejohn, 1951).

"The outstanding characteristic of the anthracotic lung is the presence of areas of consolidation, varying from deep black to dark grey, cutting with a smooth dry section like black india-rubber, situated, as a rule, at or close to the apex of the upper or the lower lobe, and often prolonged downwards as a cuirass-like sheath of dense black infiltration under the pleura. These black areas may be so dark and solid as to resemble lumps of coal or they may exhibit a fine tracery of grey lines or whorls.

Elsewhere throughout the cut surface of the lungs are seen black spots, varying in size from that of bird-shot up to buck-shot, well defined in shape as angular or polygonal maculae and standing out in sharp contrast to the chocolate-coloured or grey background of aerated lung substance.

Seen through the pleural surface of the intact lung, they are usually circular, often showing a white or grey centre surrounded by a black ring. These black spots may be few and widely separated or numerous and close together. As a rule they are more numerous in the upper portions of the lung where they may be so closely agglomerated as to be practically confluent, thus passing into the 'consolidated areas' already referred to. The consolidated areas are firm to the touch and the black spots, too, give a sensation of nodular resistance to the palpating finger as compared with the softer lung substance of the relatively unpigmented parts. The fibrotic nature of the consolidated areas and maculae is more easily appreciated by the finger than by the eye, but there may be some visible hyperplasia in and around the vessels and bronchi and along the interlobular septa.

The pleura is generally thickened and may show raised bullae or ragged patches where adhesions have been torn through or divided.

In between the black areas and maculae, the lung substance may show areas of spongy emphysema or the tissue may be wet and oedematous, varying from dark grey to haemorrhagic brown. In two cases, dying from pernicious anaemia, the lung substance was pale grey affording a marked contrast to the black areas. Towards the roots of the lungs are seen the bronchopulmonary and tracheobronchial glands, these being, in all cases, deep black in section, though sometimes marbled with fine grey streaks just as are the areas of black consolidation in the lung tissue itself.

Within the limits of this general description, there are wide variations depending upon the size and density of the blackened and consolidated areas."

These comments are particularly interesting :

"A still earlier type in which the anthracosis has stopped short at the macular stage, where the lungs, although retaining a considerable amount of dust, may be regarded as practically normal."

and again :

"In the more advanced cases of anthracosis cavitation of the apical lung tissue is frequently found and

often independently of any tuberculosis. Histological examination failed to show any tuberculosis in these lungs."

They were of the opinion that anthracosis, which they defined as a fibrosis of lungs accompanied by heavy impregnation with coal dust, was a special form of industrial disease occurring in coal miners chiefly, and to a less extent in others, who labour with coal. They were further convinced that the inhalation of silica dust was a condition essential to anthracotic fibrosis, that in fact "anthracosis is essentially a special variety of silicosis depending upon the simultaneous or subsequent accumulation of coal dust in the silica-damaged lung".

As to the effects of anthracosis on working health they observe :

"It is very important to bear in mind that in anthracosis, as in pure silicosis, simple lung fibrosis may develop to a very considerable extent without causing appreciable loss of health."

So, in effect, after 100 years, the wheel had turned full circle ; doctors were back at the beginning, again speculating, in almost identical manner, on the many problems of aetiology, pathology, course, and effects of the disease. On this occasion, however, the focus was on South Wales and not on Scotland as heretofore.

Mechanization in Coal Mines

Early in the present century there was an important development in mining methods, namely, the mechanical cutting and conveying of coal.

According to Black (1951) in 1913 only 8% of coal in Great Britain was machine cut. Lanarkshire is reported to be the home of the coal-cutter and the corresponding figure for Scotland was 22%. From this time onwards there was a general expansion of the method throughout the coalfields, though this was more rapid and more extensive in some than in others. Conveyors of bigger capacity were brought into use, while machine cutting was extended to thicker seams. In 1928, 12% of coal in Britain was mechanically conveyed. Black records that machine cutting was, until about 1930, almost entirely confined to Lanarkshire where the particular machine favoured was the disc cutter. Men still recall that in the early 1920s miners in Muirkirk (Ayrshire) used to enquire : "Who is going to Shotts (Lanarkshire) to be poisoned ?" The allusion apparently is to the fact that men returning from Shotts, where the disc cutter was in use, were noted to be short of breath when they returned to Muirkirk where the coal was hand-got.

Concurrently many pits throughout the country were becoming old, very deep, with extensive workings remote from the shaft and with the seams becoming thinner and often faulted among hard rock.

All these circumstances combined to increase dustiness of the air in the mines, not only at the coalface but generally throughout the workings.

Having regard to the scourge of the disease in South Wales during the last 20 years, it is a curious fact that mechanization was introduced much more slowly and much later in this area than in the rest of the country. There is no doubt, however, that ventilation was particularly bad in anthracite pits, partly due to the fact that the mines were becoming old and deep and also because, owing to the lack of risk of explosions propagated by coal dust, it was not so vital to maintain ventilation at such a high level as in bituminous pits where the risks of explosions were much greater.

Contributing to a debate in 1930 Mr. E. O. Forster Brown (1931), a mining engineer, related :

"We were having trouble with dust at a South Wales anthracite colliery. We were trying to overcut a seam of about 2½ feet thick above which there was inferior coal. We were cutting into the inferior coal with a chain coal-cutter and we got an enormous amount of dust. The dust was so thick that we could not see at all anywhere near the coal-cutter."

The Haldane Controversy

In the history of respiratory diseases in coal miners the year 1930 is noteworthy for the vigorous intervention of J. S. Haldane who, as a physiologist of international reputation, had made outstanding contributions to the study of respiratory function and the effects on the lungs of muscular work and mine gases (Haldane, 1916, 1918). He was mainly responsible for the introduction of stone-dusting in mines as a means of controlling underground explosions. Following his work among metalliferous miners, particularly Cornish tin miners, he was generally regarded as a leading authority on miners' phthisis or silicosis. In addition to his academic appointment as professor of physiology at Oxford, he acted as honorary director of the Mining Research Laboratory at Birmingham. From his writings and pronouncements during this period (1930 to 1939) it is difficult to avoid the impression that Haldane regarded the new outlook on miners' lung diseases as heretical and a challenge to his own work and conclusions. Besides he was plainly opposed to the trend of silicosis legislation as far as it concerned coal miners. So it emerges that his protests and arguments are characterized, not so much by cogent dispassionate

argument, but rather by advocacy in defence of his own case. It is remarkable that so great a scientist should have accepted so uncritically the mortality statistics of the Registrar General and have been so obsessed by bronchitis as a specific disabling and mortal disease. Under the circumstances it is little wonder that he came into such violent collision with clinicians and pathologists. Haldane (1931) propounded that :

"Silicosis is a disease of the lungs brought about by the inhalation of siliceous dust, and leading to tubercular infection of the lungs accompanied by great development of fibrous tissue in them. . . . Hence deaths from silicosis, when they occur, are nearly all returned as due to phthisis or tubercular lung disease."

Citing the mortality statistics of the Registrar General that the phthisis death rates for the various groups of underground workers in coal mines are substantially lower than those for all occupied and retired males he argued that, apart from workmen engaged in machine-drilling in driving roads through sandstone, coal miners did not suffer from silicosis but from bronchitis, which he attributed to heavy muscular work. Nevertheless he was compelled to acknowledge that, radiographically, changes, similar to silicosis, might exist.

"It has been shown lately that when healthy coal miners are examined with the x-rays, a good many of them present a picture which cannot be distinguished from the picture seen in the case of the Johannesburg miners. If a man in the mines at Johannesburg presented such a picture, it would be regarded as sentence of death, but this picture seems to be found in this country among coal miners, who are fairly healthy. I want to make a strong protest against the practice of diagnosing silicosis on mere x-ray examination without knowledge of the man's history and of the kind of dust he breathes."

The explanation, which he offered, was that the inhalation of coal-dust by coal miners and coal boat loaders stimulated the phagocytic activities of the lung and thus conferred a relative immunity from phthisis.

In the subsequent discussion Mr. E. O. Forster Brown (1931) made the following comment, which is remarkable in relation to the development in recent years of aluminium prophylaxis and therapy in silicosis.

"It seems to me that if the nature of that constituent in the dust could be discovered, it would go a long way towards solving this problem. I do not know how it would be applied and I should like Dr. Haldane's views on that matter, whether, if one found what the constituent was, or what mineral contained the constituent to the maximum percentage, some spraying of the noxious dust during boring operations by means of this other dust could be evolved? Would it be practical politics to treat men with inhalations containing this constituent,

supposing it could be isolated, before they went in to drive hard measure drifts in a rock with a high free silica content? If something of this kind could be done it might solve the whole problem."

Haldane replied:

"It seems to me that the inhalation of dust of any kind is bound to cause trouble when it is thick enough; and anthracite-dust seems to be worse than that from other kinds of coal. Whatever the constituent in coal-dust is that stimulates elimination of the dust it must be applied along with the dust, and in a particular form and some sort of proportionate amount."

He emphasized the unduly high mortality from bronchitis in coal miners and reiterated that it was due to heavy muscular work and not primarily to the noxious effects of dust. He did, however, state that "it seemed practically certain that excessive inhalation of coal-dust or shale-dust must cause bronchitis and ought therefore to be avoided".

Quoting from the statistics of the Registrar General that the comparative mortality from bronchitis in 1921 to 1923 for miners (hewers and getters) varied between the extremes of 33.8 for Leicestershire and 139.3 for South Wales, as compared with 49.6 for all occupied and retired males, Collis (1931) argued that, as the physical exertion in these mining areas probably approximated closely, these figures did not support the muscular exertion theory.

Until his death in 1936 Haldane persisted, with little alteration, in his views and his final contribution, largely a restatement of his researches and a justification of his conclusions, appeared posthumously (Haldane, Haynes, Shaw, and Graham, 1939). Thus the article records:

"X-ray appearances similar to those of distinct silicosis have been found by the investigation of the King Edward VII Welsh National Memorial Association to be present in a large proportion of the lungs of colliers, who are at their ordinary work, and have worked for many years on the coal-face exclusively, and never in stone drifts where they might be exposed for long periods to dust from highly siliceous rock. These men, however, are not subject to any excess of phthisis, as shewn clearly by the occupational mortality statistics of the Registrar General, so that to regard their x-ray appearances as evidence of silicosis seems quite inadmissible. The other fact is that when a man is suffering from either bronchitis or ordinary phthisis the local means of getting rid of inhaled dust is partly paralysed. Hence dust accumulates in excessive quantity in certain parts of his lungs. This, together with the fibrosis and x-ray appearances to which it apparently gives rise, is not due to silicosis, but is a secondary result of the bronchitis or phthisis."

There can be little dispute that, in relation to bronchitis, Haldane's intransigence beguiled him, but, this notwithstanding, his vital contributions

to silicosis rank among the most significant in the world literature. Furthermore, present experience of severe disability due to emphysema without evident pneumoconiosis, occurring among machine-men at the coal-face and among labourers in heavy foundries, may yet lead us to re-examine Haldane's work on muscular exertion in relation to bronchitis.

To the very end of his life Haldane insisted that, notwithstanding similar radiographic appearances, silicosis and pneumoconiosis of coalworkers were distinct pathological conditions. This contention has since received considerable support from the recent researches of Gough (1944, 1947) and of Heppleston (1951).

The Tuberculosis Riddle

So it appears that, by 1930, the lung diseases of coal miners were again compelling attention, particularly in South Wales. While doctors were investigating the clinical and pathological aspects, the miners, through their trades unions, were concerned with the associated problems of workmen's compensation.

Among experts, all over the world, it was accepted that tuberculosis of the lungs was the most serious risk of those who work in dusty occupations, this association being peculiarly intimate between phthisis and crystalline silica. Against this coal miners seemed to be exceptional, for while it had been established that silicosis did occur among men occupied in certain operations in coal-mines, the incidence of pulmonary tuberculosis among coal miners was considered to be less common than among comparable age groups of males in the general population. Commenting on this apparent anomaly a leader writer (*Lancet*, 1932) pertinently observed:

"In point of fact there is evidence that in certain coalfields, at any rate, this freedom from tuberculosis does not obtain; and it is possible that the data upon which the statistics relating to the industry as a whole have been based have been accepted without sufficient scrutiny."

However, this comparative freedom of coal miners from tuberculosis was the subject of a series of scientific studies (Cummins and Weatherall, 1931, 1933; Cummins and Williams, 1938) from which it was suggested that

"the coal miner may be protected to some extent from pulmonary tuberculosis by the power of the coal dust, with which his lung is saturated, to adsorb the tuberculin of the tubercle bacillus, so that the clinical manifestations of the disease are modified and, in the absence of a post-mortem examination, death may be wrongly certified as due to bronchitis or uncomplicated silicosis."

Sericite

The discovery that asbestos, a silicate, could produce disabling and fatal fibrosis of the lungs, had done little to displace free crystalline silica from its supreme position as the cause of pneumoconiosis, but the whole problem of aetiology was re-opened in 1933 when W. R. Jones, a geologist, propounded his thesis about sericite as the causative agent in silicosis.

Using the petrological microscope to investigate a series of human lungs, he related the mineral residues, which he identified in the lung ash, to the geological constitution of the materials which had caused the disease. He (Jones, 1934) concluded :

"The outstanding fact should, however, be emphasized that in all the mineral residues of these silicotic lungs, there are countless mineral fibres, hundreds of which are present for every grain of quartz that can be recognized."

and

"The mineral residues obtained from the 29 silicotic lungs of employees who had been engaged in the various industries in this country consist, therefore, mainly of myriads of minute acicular fibres of sericite."

Sericite is a fibrous mineral silicate of aluminium and potassium mainly produced by alteration of feldspar, when it is subjected to great geological pressure. It exists either as minute scales or fine, needle-like fibres. Dr. Jones, while admitting that, in exceptional cases, free silica might be the causal agent, attributed silicosis to the acicular form of sericite.

Contrasting the serious incidence of the disease in the South Wales coalfield with its almost complete absence in Scotland, Jones explained that "the Scottish sandstones contain very few such fibres, and, indeed, compared to the myriads present in the South Wales rocks, fibres of sericite in these Scottish rocks are rare".

All over the world pathologists and mining engineers, while making some critical reservations, acclaimed the author and the hypothesis. Haldane (1934), as appears from this report of his contribution to the discussion, remained adamant and unconvinced :

"It seemed to him that evidence was constantly accumulating that scattered fibrosis, giving essentially the same x-ray picture as real early silicosis, was comparatively common as a result of excessive dust inhalation, but without accompanying liability to tubercular infection. He did not think that this condition might be called silicosis, and so far as the evidence went, it seemed to arise *from excess of any kind of dust*, and not merely of silicate dust, so that *pneumoconiosis* seemed the most suitable name for it" (italics inserted).

This hypothesis has not withstood the critical arguments of experts, nor has it been confirmed by animal experiments. Moreover, certifications in recent years have revealed that many mining areas outside South Wales produce numerous cases of the disease.

The Beginning of the Modern Phase in South Wales

In 1931 the Various Industries (Silicosis) Scheme, 1928, was amended and extended, but so far as coal miners were concerned the only change was that compensation became payable in respect of partial disablement. This means that the provisions of the scheme were still restricted to workmen employed in drilling and blasting in silica rock. Ultimately it was proved that this restriction constituted a serious hardship and so in 1934 the scheme was amended to include all underground colliery workers.

During the early 1930s certifications among coal miners under the Various Industries (Silicosis) Scheme, 1931-34, by the Silicosis Medical Board, focused attention on the progressively mounting number of cases of silicosis, which were occurring almost entirely in South Wales and predominantly in the anthracite area around Swansea. This narrow geographical concentration of cases was impressive and not a little puzzling. Harper (1934), a radiologist practising in the South Wales coalfield, wrote "silicosis is the fashionable disease in this area at present". He described the system, which enabled a radiographic examination to be made in practically all miners in the neighbourhood.

"There seems to be little mystery why there are so many cases of silicosis in this part (Swansea area) of South Wales when the full facts are known. . . . It is probably safe to state that more colliery workmen have been x-rayed in this area than in any other part of the coalfield ; hence more cases of silicosis have been found than in any other areas where no such examination has been carried out. This, it would seem, is part of the reason why there are many more known cases in this area than in any other part of the country. What the results of similar examinations would yield as regards silicosis in the other coalfields it is impossible to state until such examinations are carried out."

The following year the same writer (Harper, 1935) directed attention to the occurrence of similar lung disease among colliery surface workers who had never worked underground. He again observed :

"These findings would also substantiate with some little reason my previous statement that the extent of the disease in the steam and soft coal areas is absolutely unknown, since no systematic examination has been carried out in these areas."

Fisher (1935), H.M. Medical Inspector of Mines, ultimately presented the situation in precise figures derived from official sources.

"Further, it soon became apparent that an interesting grouping of cases was taking place. The number from the Somerset area hardly increased, and very few cases were being reported from any part of the country except South Wales. For instance, out of 81 cases certified by the Medical Boards as dying of silicosis up to December 31st, 1933, 69 occurred in South Wales (29 in the Cardiff division and 40 in the Swansea division). Out of 257 total disablement cases, 233 were certified in South Wales (92 in Cardiff division and 141 in the Swansea division). We therefore see that out of 449 certificates issued, 398 were to South Wales cases (in 17 cases 2 certificates were issued in respect of each man—one for disablement and later another for death)."

Referring to the practice of stone-dusting as a factor in the causation of the disease Fisher emphasized that many cases occurred in anthracite mines, where no stone-dusting was done. He also demonstrated that there was no correlation between silicosis and the amount of drilling.

"While South Wales as a whole accounts for only one-quarter of the total number of picks and drills used for purposes other than getting coal, nearly 90% of the cases of silicosis occurred in this district. The position in the Swansea division is even more striking, the corresponding figures being, picks and drills 5% and silicosis cases 54%."

In the discussion Cummins (1935) contributed this important clinical observation :

"It cannot be too much stressed that this pneumoconiosis of coal miners is a *progressive* condition, gradually passing on from its harmless early stages, through a condition of some respiratory embarrassment still consistent with working health, until it finally attains, in those persons most seriously affected, to a condition of dyspnoea and breathlessness which makes work impossible."

and again :

"It is a great mistake to suppose, as do those who pin their faith on the Registrar General's statistics, that this form of coal miners' pneumoconiosis is exempt from the risk of being complicated by tuberculosis."

There was an apparent shift of emphasis from silicosis to the more general term pneumoconiosis as revealed by Owens (1935) who remarked that "it would be better if we dropped the term 'silicosis' in favour of koniosis, as this simply means a dust disease and does not suggest the specific dust; pneumoconiosis or pneumoconiosis are both too cumbersome."

Even Collis (1935) was led to comment that "some of the anomalies were due to gathering under the head of silicosis more than one pathological condition."

Haldane (1935) again rose in defence and offence :

"Let me say at once what I believe these cases to be. I think these cases are primarily cases of either bronchitis or ordinary phthisis, the collections of dust with such extra fibrosis as is found on post-mortem or x-ray examination being due to paralysis by bronchitis of the normal process of dust elimination. This normal process and the development of fibrous tissue which results when it is either overwhelmed or fails in any other way have been followed carefully in the series of animal experiments carried out by Beattie and Mavrogordato, Carlton and Haynes.

"Nevertheless, this fibrosis cannot be identified with silicosis unless it occurs in the whorled form characteristic of silicosis, or is accompanied in man by the liability to tubercular infection which is characteristic of silicosis, and makes silicosis such a dangerous condition."

To the layman medical opinion appeared in conflict, so much so that Dr. William Cullen (1935), a mining engineer, felt impelled to say that "in his opinion the mining man could forget about the medical man for the present. His job was to get rid of the dust by all and every means in his power".

So far references, in the main, have been to individual experience and opinion. No general view of the situation which had developed became available until 1938, when the Registrar General published the occupational mortality statistics covering the years 1930-32. In the discussion of the mortality of coal miners from all causes, these observations appear significant :

"The lack of improvement during the last decade in the rates at ages between 20 and 55 contrasts with a considerable decline in the general mortality rates of males; the position of coal miners' mortality figures in relation to those of all occupied and retired males deteriorated considerably between 1910-12 and 1930-32 at each age period between 25 and 55, that is to say, although there was a fall in their death rates during the 20 years the *improvement was apparently much less* (italics inserted) than in the general population and was confined to the first half of the interval."

As to respiratory diseases in particular :

"At ages under 55 the respiratory tuberculosis rates still remained below, but approached nearer to, the average rates, whilst the non-tuberculous respiratory death-rates, notwithstanding an absolute improvement since 1921-23, reached levels about 20% in excess of the average."

The inference was clear, namely that coal miners, for some unexplained reason, were not benefiting from public health measures to the same extent as other groups; respiratory diseases were on the increase in this occupation.

Meanwhile in 1934, arising out of their duties of certification under the Various Industries (Silicosis) Scheme, 1931-34, Keating and Thomas,

members of the Medical Board for Silicosis in South Wales, directed attention to a type of case frequently encountered among claimants in this area. They reported (quoted by McVittie, 1950) that many cases occurred in which the diagnosis was difficult because the industrial history and clinical and morbid anatomical findings led to the conclusion that a pathological condition of the lungs had developed which, while not characteristic of classical silicosis, closely simulated silicotic fibrosis. In the great majority of these cases, the radiograph displayed abnormal appearances in the lungs, which, however, did not conform to those generally accepted as indicative of silicotic nodulation. The films were divisible into three groups representing: (a) emphysema only; (b) reticular fibrosis of a coarse type; and (c) reticular fibrosis of a fine type, more marked in mid-zone.

In 1936 Dr. Charles L. Sutherland, Chief Medical Officer of the Silicosis Medical Board, directed the attention of the Home Secretary and of the members of the Industrial Pulmonary Diseases Committee of the Medical Research Council to the fact that, in the previous three years, claims for compensation on account of silicosis by coal miners had increased by 70% and these were almost entirely in South Wales (McVittie, 1950). He also recorded that the number of certificates refused in the same period had increased by 300%. The great majority of these refused cases belonged to the anomalous types described by Keating and Thomas. Disabled workmen and their dependants, who had failed to obtain compensation, not unreasonably felt aggrieved. The situation demanded immediate scientific investigation.

Chronic Pulmonary Disease in South Wales Coal Miners

So it came about that in 1936 the Medical Research Council were asked by the Home Office and the Mines Department to investigate the problem of chronic pulmonary disease among coal miners, with particular reference to conditions in the South Wales coalfield. The Council undertook to do this. The inquiry which was set up extended over the years 1937-42 and consisted of a medical survey and pathological and environmental studies. Three reports (Medical Research Council, 1942, 1943, 1945) were issued and these presented an up-to-date, comprehensive, authoritative statement of the position as it existed in South Wales.

Hart and Aslett, the leaders of the medical investigation, confirmed that hard-heading workers,

driving through rock containing a high proportion of quartz, were subject to a special and well-recognized risk of developing silicosis but these workmen were very few and provided only a small fraction of the cases of pulmonary abnormality. The overwhelming incidence of cases occurred in colliers working at the coal-face and to a less extent among other underground workers. Similar lung changes were discovered amongst surface workers on the screens and coaltrimmers at the docks. Indeed it was this observation which underlined the importance of coal dust and not rock dust as the causative agent of pneumoconiosis of coal workers.

Rank of Coal Hypothesis

It appeared that the incidence of the disease was not uniform throughout the coalfield. Thus it was high in the anthracite or hard coal area, low in the bituminous or soft coal area, with semi-bituminous or steam coal mines occupying an intermediate position. These findings induced Hart and Aslett to suggest that the incidence of the disease was in some way related to the rank of coal that was mined. According to their report the character and rank of coal in the South Wales coalfield change gradually from north-west to south-east. The highest rank of anthracite with about 5% of volatile matter is found in the north-west, and, proceeding towards the south-east, the volatile content steadily increases, the coal changing in character to lower-rank anthracite, steam coal, and finally, in the south-eastern and eastern parts of the coalfield, to bituminous coal with up to 36% volatile matter.

Thus according to Hart and Aslett the nature of the coal as represented by "rank" was the chief factor in determining the varying incidence of the disease in separate areas of the coalfield. They did not accord to the concentration of airborne dust the importance which later investigators have attributed to it.

Reticulation

The pathological changes in the lungs were reflected by radiographic appearances, which the authors classified as (a) reticulation, (b) nodulation, (c) coalescent nodulation, (d) massive shadows, and (e) multiple fluffy shadows. Nodulation and massive shadows were already well-recognized as features of silicosis. Reticulation, however, was a new term used to denote the radiographic appearances in which the lung fields were more or less diffusely altered by fine, net-like shadows. It was the earliest radiological sign of any abnormal changes

in the lungs and was the commonest x-ray abnormality amongst the younger colliers in both anthracite and non-anthracite mines. Thus Hart and Aslett had defined a new concept if not a new variety of pneumoconiosis.

"Reticulation is an earlier x-ray change than the better recognized discrete nodular and conglomerate shadows, and in films showing these latter appearances it is generally visible in addition to the more noticeable consolidation; the evidence further suggests that it represents an essential part of the pneumokoniotic process leading to consolidation, i.e., that it is a pre-stage of the latter group of changes."

and

"Even when reticulation is not accompanied by clinical disability, its presence still implies the risk—considerable in high incidence mines—of the later progression to consolidation with its more serious accompaniments."

Dust Reticulation

Belt and Ferris (M.R.C. Special Report Series, 243) studied the pathology of the lung lesions and recorded that:

"While the word reticulation has come into the terminology of pneumoconiosis via radiological channels, it is, by coincidence also applicable as a descriptive term to the corresponding histological changes, though for a somewhat different reason, for here the network to which the term applies is a microscopic structure composed of dust-laden cells and their connecting fibres. The dust-laden tissue in these cases is, in fact, nothing else than reticular tissue as defined by Maximow and Bloom (1938)."

These histological changes, which they described in detail and to which they gave the name dust-reticulation, were entirely different from nodular collagenous fibrosis of silicosis.

"The condition is so well-known under the mistaken designation of simple anthracosis that a detailed account of it may seem superfluous. Even at the risk of repetition, however, we should like to describe the condition carefully, because it seems desirable that it should be reconsidered in the light of certain new findings which point to its being more in the nature of a disease than was formerly thought."

The general characters of the lesion are best seen in large histological sections (Christeller technique) under low power magnification. For this purpose the sections may be mounted on lantern slides and examined to good effect by simply projecting them on to a screen. The dust deposits are scattered in a lace-like pattern of fine streaky processes throughout both lungs, in a manner corresponding to the diffuse lattice-work shadows seen by x ray. The change is diffuse and symmetrical, affecting all parts of the lung more or less uniformly, with the exception of the extreme base, where it is often less marked than elsewhere. The distribution is essentially that of the lymphatic pathways and depots. The root glands are packed with dust, while in the lung itself there is a heavy storage along the interstitial tracts, in the perivascular and peribronchial sheaths (particularly

the former), as well as in the interlobular septa and subpleural tissues; often enough, moreover, there is some extension even into the interalveolar septa. Under high power, the collections are distinctly patchy, with a notable tendency towards localized concentrations. The appearance may be likened to that of innumerable small dust-ridden cobwebs, all linked up with each other, more or less in continuity, or in series. They represent traps where dust-laden phagocytes have been brought to rest and transformed into fixed-tissue phagocytes which then become knit together into a meshwork by the elaboration of reticulum fibres."

So in effect a new type of pneumoconiosis was established; the characteristics were radiographic reticulation and histological dust-reticulation. As will appear these concepts were later to be opposed and supplanted.

The research team had identified radiographic and pathological changes, which could not rightly be designated silicosis, so it became necessary to invent a new name. Hart and Aslett argued that "until the causes of the various lung changes due to dust in the coal-mining industry were more fully established, some general term should be used for legal purposes to cover both reticulation and consolidation". They suggested "pneumoconiosis of coalminers" which the Committee on Industrial Pulmonary Disease later altered to "pneumoconiosis of coal-workers" so as to include workers engaged in any operation underground in coal mines, in screen workers at collieries, and in coal trimmers at docks.

Following the issue of the report, the legislature by the Workmens Compensation Act, 1943, gave partial effect to these recommendations in so far that the disease for which compensation became payable was "pneumoconiosis" meaning "fibrosis of the lungs due to silica dust, asbestos dust, or other dust and includes the condition of the lung known as dust reticulation".

The immediate result, as shown by Table 1 (McVittie, 1949), was a tremendous increase in the number of compensation claims and certifications among coal miners.

It should be pointed out that the peak figure of 5,224 for Wales in 1945, while administratively accurate, is misleading. In 1944 the medical boards at Cardiff and Swansea were overwhelmed by claims so that examinations and certifications were often over one year in arrears. This means that a substantial number of the certifications in 1945 resulted from applications made in 1944.

Commenting on the situation which had developed Fletcher (1948) said:

"The total number of certified cases in South Wales is approximately seven times the number

TABLE 1

NEW CASES CERTIFIED BY THE SILICOSIS MEDICAL BOARD IN GREAT BRITAIN DURING THE YEARS 1939 TO 1947*

Region	1939	1940	1941	1942	1943*	1944	1945	1946	1947
Scotland	7	2	3	6	10	109	148	166	322
Northern	2	5	2	4	12	79	57	71	93
North-Eastern	6	7	8	6	25	85	95	127	155
North-Midland	—	1	—	1	4	10	13	25	17
North-Western	10	10	8	7	27	53	81	97	136
Wales	418	448	499	763	1,155	1,608	5,224	3,804	2,867
Midland	12	11	13	24	48	95	132	99	110
Kent	10	—	2	10	25	30	71	51	79
Total	465	484	535	821	1,306	2,069	5,821	4,440	3,779

* Coal Mining Industry (Pneumoconiosis) Compensation Scheme began on July 1, 1943.

in the rest of the country. Since there are nearly six times as many coal miners in the rest of the country, the incidence of certified disease is nearly 40 times greater in South Wales."

He considered that the main causes of this increase since 1943 were (a) the acceptance of the radiological condition of reticulation; (b) the operation of the Essential Works Order; and (c) more general awareness of the disease and intensive mechanization introduced in the east Glamorgan pits.

McVittie (1949) attributed the alarming figures in South Wales to a mass radiographical investigation, whereby the official medical boards were used not only for certification but to provide an irregular system of periodical radiographical examinations of coal miners, fit and disabled.

More recent experience is provided by estimates compiled by the Ministry of National Insurance from official records for the period April–December, 1950, shown in Table 2.

It should be realized that certifications for the purposes of workmen's compensation are not a true measure of the incidence of pneumoconiosis in the different mining divisions. McVittie (1949) states:

"Experience has proved that the evidence of the disease in any industry is proportionate to the number of x-ray examinations made of the workers exposed to dusts."

This is also true of the separate geographical

divisions of an industry, and, as already recorded, mass radiography in the mining industry has until now been almost entirely restricted to South Wales.

Employment Problems of the Disabled

Reference has just been made to the great increase in claims for compensation which followed the new legislation for pneumoconiosis of coal miners. In view of the preceding events this was not surprising, and, what is more, it was already clear that serious related problems of social and economic importance to the individual and the nation were developing concurrently. These arose from the fact that the award and acceptance of compensation for the disease involved immediate and permanent suspension from practically all work in the mines and, in effect, from all occupations involving exposure to noxious dust. Accordingly increased certifications meant a progressively larger number of partially disabled miners in need of alternative work.

So on May 21, 1943, just a few weeks before the Coal Mining Industry Pneumoconiosis (Compensation) Scheme became effective on July 1, the Minister of Fuel and Power appointed a committee:

"To advise as to measures which should be taken to provide for the medical treatment and rehabilitation treatment or otherwise, of coal miners in the Wales Region suffering from Pneumokoniosis and to make recommendations as to what should be the

TABLE 2
PNEUMOCONIOSIS IN BRITISH COAL MINERS IN 1950

Area	Working Population (1000s)	Certified Cases (Annual Rate per 1000)	Claims (Annual Rate per 1000)
Wales, Monmouth, Forest of Dean, Bristol, and Somerset	105.9	18.91	32.58
Kent	6.0	14.20	18.00
N. Staffordshire, S. Staffordshire, Cannock, Shropshire, and Warwick	54.5	5.33	6.53
Scotland	80.8	5.21	7.33
Lancashire and North Wales	56.3	3.60	6.04
Durham	106.8	2.19	3.40
Yorkshire	134.0	1.79	3.04
Northumberland and Cumberland	48.7	0.54	1.23
Nottinghamshire, Derbyshire, and Leicester	94.7	0.31	0.51

lines of initial action and of concurrent investigation and study to provide a basis for further development."

The committee's report was published just one year later. They recorded that, after giving the fullest consideration to the evidence and information which they had received, they were of opinion that there was insufficient knowledge available to enable them to advise or recommend *large scale* measures of treatment including rehabilitation.

Having regard to succeeding events the following recommendations are noteworthy. (1) The early establishment of a treatment and rehabilitation research centre with accommodation for 30 in-patients, equipped with facilities for clinical study and research; (2) the provision of facilities for pathological research into the early dust changes in the lung, the progression of the lesion, and the part played by tuberculosis and other infections. Where possible the pathological findings should be correlated with x-ray findings. These recommendations were duly implemented in 1945 by the establishment of the Pneumoconiosis Research Unit at Cardiff under the direction of Dr. Charles M. Fletcher.

Meanwhile a working party under the chairmanship of Mr. D. R. Grenfell, M.P., was set up to investigate the problem of providing work in South Wales for persons suspended from the mining industry on account of silicosis and pneumoconiosis. They advised the establishment of new light industries in certain of the most seriously affected colliery areas and expressed the opinion that "the finding of new employment for pneumoconiotics would not present great difficulty, if full employment could be ensured in the areas concerned."

Experience soon proved that this view was over-optimistic. In 1951 in reviewing the "Social Consequences of Pneumoconiosis among Coal-miners in South Wales," Hugh-Jones and Fletcher recorded that "at present some 5,000 men with pneumoconiosis, three-quarters of whom are probably capable of work under normal industrial conditions, are unemployed."

Moreover, this happened despite the fact that as from July, 1948, workmen were allowed to obtain compensation while continuing at work in the mines. Indeed so acute did this problem of unemployment ultimately become that in June, 1951, the law was further amended so that:

"Men suspended under the Workmen's Compensation Acts because of pneumoconiosis or silicosis, unaccompanied by tuberculosis, may now take up employment in the coal-mining industry if they are passed by a pneumoconiosis medical board as fit to do so."

The authorities never explained whether this complete alteration of attitude to the continued employment of affected workmen in the mines was justified on medical grounds, or by the reduction of dust in the mines, or whether it was simply an expedient to mitigate the disaster of unemployment among the disabled.

Pathological Studies

As recorded, Belt and Ferris (1942) had presented their concept of the minute pathology of coalworkers' pneumoconiosis under the term dust reticulation. Almost immediately afterwards their work was superseded by the researches of Gough (1944, 1947) of the Welsh School of Medicine at Cardiff, who, using the new technique of large tissue sections (Gough and Wentworth, 1949; Gough, James, and Wentworth, 1949) submitted that:

"The earliest lesion in the lung due to the inhalation of coal dust as such is the coal nodule; it is present in miners and coal trimmers. In and around the coal nodules, which in reality are collections of coal dust, is developed a peculiar type of emphysema which is focal in nature."

Heppleston (1947), working in the same laboratory, later gave a detailed account of the development of the coal nodule and of his views on the mechanism of the production of focal emphysema.

More recently Heppleston (1951) has presented evidence that the essential lesion of coal pneumoconiosis is the same in coalworkers from coalfields in all parts of the world and he again asserts the clear distinction between coal pneumoconiosis and silicosis. Moreover, he concludes that it is a quantitative effect:

"Simple mechanical accumulation of dust, rather than the activity of free silica, is the main factor in the genesis of simple pneumoconiosis in coal workers. There is no evidence of the operation of any agent other than dust."

The coal nodule is now accepted as the specific lesion but there is still controversy about the mode of development of focal emphysema (McVittie, 1950).

Gough (1947) also concluded that pneumoconiosis of coalworkers was not one but two diseases. The first, simple pneumoconiosis, was caused by the inhalation of coal dust which accumulated in small foci, where it excited a minimal amount of reticulin fibrosis. The second, infective pneumoconiosis, was due to the combined action of coal dust and tuberculous infection, whereby "infective nodules" composed of collagenous fibrous tissue developed and later aggregated to form areas of massive fibrosis.

Some support for this view of infective fibrosis was provided by the bacterial studies of Rogers (1946) and by the observations of Stewart (1948) on erythrocyte sedimentation rates. The problem is at present being studied by a mass radiographic examination of the whole population of the Little Rhondda Valley.

Recent Researches

Since the Pneumoconiosis Research Unit was instituted at Cardiff in 1945, Dr. Fletcher and his colleagues, as a result of extensive researches, have made many contributions to the literature of pneumoconiosis, particularly as it affects coal miners in South Wales.

Davies and Mann (1949) consider that reticulation should be abandoned as a term descriptive of radiographic changes. They submit that the characteristic early x-ray appearances are minute opacities, which, pathologically, appear as small foci of dust deposition, associated with reticulin or occasionally collagenous fibrosis, throughout the lung; the coal nodules of Gough.

"The characteristic opacities are minute (0.5-1.5 mm. diam.), more or less circular and fairly well-defined in outline. They are usually arranged in clumps. The appearance is thus of a granular type, but fine linear opacities often connect adjacent minute opacities, enclosing small, translucent areas which results in a fine lace-like appearance."

This compares with the original description of reticulation (Hart and Aslett, 1942);

"Lung fields show a fine network sometimes sharp and lace-like in pattern, but much more often blurred in appearance."

It is generally agreed that it is not possible radiographically to identify either the presence or extent of focal emphysema.

The Unit have also concluded that the radiographic appearances may be divided into two main forms, simple pneumoconiosis and progressive massive fibrosis, corresponding with the pathological distinction made by Gough.

In simple pneumoconiosis "the first radiological abnormalities to appear are minute opacities up to 1 mm. in diameter; these increase in number and size, and to a certain extent in radiological density, until the whole of both lung fields is thickly covered with opacities ranging up to 3 or 4 mm. in diameter, which are usually composed of aggregates of minute opacities".

Progressive massive fibrosis consists of localized, more homogeneous opacities of longer size, which commonly appear in the upper zones, particularly on the right side. This appearance is always accompanied by some degree of simple pneumo-

coniosis and the combination has been named complicated pneumoconiosis.

In the radiological diagnosis of simple pneumoconiosis Fletcher and Oldham (1949) emphasized the errors, made by the same observer and between different observers, which occur in the recognition and classification of the early categories. To minimize these errors and so achieve greater consistency they advocated the use of standard reference films. In a special test (Fletcher and Oldham, 1951) this method was found to have some value and an effort is now being made to have it adopted internationally (Fletcher, 1950; Cochrane, Davies, and Fletcher, 1951).

The course of the disease under various environmental conditions is of considerable importance in prognosis and in deciding whether or not a patient may safely continue in his occupation. This aspect has been studied by several workers (Williams, 1933; Stewart, 1948; Davies, Fletcher, Mann, and Stewart, 1949). Moreover, in this problem Gough's assertion that pathologically there are two distinguishable disease processes, simple pneumoconiosis and infective pneumoconiosis, is peculiarly relevant. Radiographically the former is characterized by scattered minute opacities and the latter by massive shadows. A further relevant point is that (Cochrane, Fletcher, Gilson, and Hugh-Jones, 1951):

"On the average, young men even with quite advanced simple pneumoconiosis are not seriously disabled. Men with progressive massive fibrosis, on the other hand, are usually quite markedly disabled and their disability increases both with age and the radiological degree of the progressive massive fibrosis."

According to Davies and his colleagues (1949) simple pneumoconiosis, judged radiologically, does not progress *as such* in the absence of further coal dust inhalation. With regard to massive fibrosis, however, the situation, as summarized by Cochrane and others (1951) is rather different:

"(1) If a man is removed from further exposure to dangerous dust when he has only a small amount of simple pneumoconiosis, he is most unlikely to develop progressive massive fibrosis; (2) once a man has enough simple pneumoconiosis, progressive massive fibrosis may develop with equal likelihood whether or not dust exposure ceases; (3) once massive fibrosis has started it is nearly always progressive, though at widely varying rates."

These observations led Fletcher (1950) to suggest that, radiologically, there is a "critical stage" of simple pneumoconiosis at which liability to progressive massive fibrosis arises, and so he argued that if men could be removed from the danger before this stage was reached their future health

would be safeguarded. To fulfil this desirable purpose a system of periodic radiographic examinations of coal workers was advocated (Fletcher, 1948; Cochrane, Fletcher, Gilson, and Hugh-Jones, 1951). This programme, if implemented, would assist to mitigate the evils of the disease in the individual and in the community.

Despite all these researches the fundamental problems of aetiology and pathology remain unresolved. It is, however, agreed that mine dust (a more general term than coal dust) is an important factor in producing the disease.

So recently as 1948 H.M. Chief Inspector of Mines (Bryan, 1950) recorded :

"There is no doubt that one result of the adoption of many of the present methods of machine mining is that the production of dust in mines has increased in recent years and is still increasing. If we are to get rid of the scourge of pneumoconiosis, this process must be reversed."

Mining engineers have already made great advances in the control of air-borne dust, notably in South Wales by water infusion of the solid coal at the working face before actual coal-getting begins. The National Coal Board have now established a full-time comprehensive occupational health service, covering every pit throughout the country. This service is in future to be assisted by advisory panels of experts on epidemiology, psychology, physiology, and industrial medicine (*Lancet*, 1952). In addition the Inspectors of Mines continue to ensure the observance of mining regulations.

The future chapters of this story will be written by the achievements of the team, research workers, engineers and doctors, supported by management and workmen. The cost of conquest of the disease, in money, will be great; the price already paid, in human suffering, has been too great.

I am deeply grateful to all the authors whose published work has enabled me to compile this short history. It has not been possible to notice every contribution but in the selection of material and in such comments as I have interpolated, I have tried to be completely objective.

REFERENCES

- Belt, T. H., and Ferris, A. A. (1942). *Spec. Rep. Ser. med. Coun. Lond.*, No. 243.
- Black, J. (1951). Thesis for degree of M.D., University of Glasgow.
- Brown, E. O. Forster (1931). See Haldane, J. S. (1931), p. 426.
- Bryan, A. M. (1950). *Report of H.M. Chief Inspector of Mines for the Year 1948*. H.M.S.O., London.
- Cochrane, A. L., Davies, I., and Fletcher, C. M. (1951). *British Journal of Industrial Medicine*, 8, 244.
- , Fletcher, C. M., Gilson, J. C., and Hugh-Jones, P. (1951). *Ibid.*, 8, 53.
- Collis, E. L. (1931). See Haldane, J. S. (1931), p. 443.
- (1935). See Fisher, S. W. (1935), p. 398.
- , and Gilchrist, J. C. (1928). *J. industr. Hyg.*, 10, 101.
- Cullen, W. (1935). See Fisher, S. W. (1935), p. 399.
- Cummins, S. L. (1927). *J. Path. Bact.*, 30, 615.
- , and Sladden, A. F. (1930). *Ibid.*, 33, 1095.
- , and Weatherall, C. (1931). *J. Hyg., Camb.*, 31, 464.
- , — (1933). *Ibid.*, 33, 295.
- , and Williams, E. M. (1938). *Ibid.*, 38, 638.
- Davies, I., and Mann, K. J. (1949). In *Proc. 9th int. Congr. industr. Med., London*, 1948, p. 768. Bristol.
- , Fletcher, C. M., Mann, K. J., and Stewart, A. (1949). *Ibid.*, p. 773.
- Fisher, S. W. (1935). *Trans. Instn Min. Engrs, Lond.*, 88, 377, 409.
- Fletcher, C. M. (1948). *Brit. med. J.*, 1, 1015, 1065.
- (1950). *Proceedings of the Third International Conference on Pneumokoniosis*, Sydney, 1950. International Labour Office, Geneva. (In the press.)
- , and Oldham, P. D. (1949). *British Journal of Industrial Medicine*, 6, 168.
- , — (1951). *Ibid.*, 8, 138.
- Gough, J. (1944). *Report of the Advisory Committee on the Treatment and Rehabilitation of Miners in the Wales Region Suffering from Pneumokoniosis*, p. 17. H.M.S.O., London.
- (1947). *Occup. Med.*, 4, 86.
- , James, W. R. L., and Wentworth, J. E. (1949). *J. Fac. Radiol. (Lond.)*, 1, 28.
- , and Wentworth, J. E. (1949). In *Proc. 9th int. Congr. industr. Med., London*, 1948, p. 661. Bristol.
- Haldane, J. S. (1916). *Trans. Instn Min. Engrs, Lond.*, 51, 469.
- (1918). *Ibid.*, 55, 264.
- (1931). *Ibid.*, 80, 415.
- (1934). See Jones, W. R. (1934), pp. 387-91.
- (1935). See Fisher, S. W. (1935), pp. 388-90.
- , Haynes, F., Shaw, A., and Graham, J. Ivon (1939). *Trans. Instn Min. Engrs, Lond.*, 97, 40.
- Harper, A. (1934). *Brit. med. J.*, 1, 920.
- (1935). *Ibid.*, 1, 1264.
- Hart, P. D'A., and Aslett, E. A. (1942). *Spec. Rep. Ser. med. Res. Coun. Lond.*, No. 243.
- Heppleston, A. G. (1947). *J. Path. Bact.*, 59, 453.
- (1951). *Arch. industr. Hyg.*, 4, 270.
- Jones, W. R. (1933). *J. Hyg., Camb.*, 33, 307.
- (1934). *Trans. Instn Min. Metall., Lond.*, 43, 341.
- Keating, N., and Thomas, R. W. Quoted by McVittie, J. C. (1950).
- Kemp, F. H., and Wilson, D. C. (1946). *Lancet*, 1, 172.
- McVittie, J. C. (1949). *Postgrad. med. J.*, 25, 618.
- (1950). *Proceedings of the Third International Conference on Pneumokoniosis*, Sydney, 1950. International Labour Office, Geneva. (In the press.)
- Meiklejohn, A. (1951). *British Journal of Industrial Medicine*, 8, 127.
- Middleton, E. L. (1920). *Tubercle (Lond.)*, 1, 257.
- Owens, J. S. (1935). See Fisher, S. W. (1935), p. 403.
- Rogers, E. (1946). *Lancet*, 1, 462.
- Stewart, A. (1948). *British Journal of Industrial Medicine*, 5, 120.
- Summons, W. (1907). *Miners' Phthisis at Bendigo*. Stillwell and Co., Melbourne. Quoted by Collis, E. L. (1951). *Industrial Pneumoconiosis, with Special Reference to Dust-Phthisis*. Milroy Lectures, 1915, H.M.S.O., London, 1919.
- Sutherland, C. L. Quoted by McVittie, J. C. (1950).
- Tattersall, N. (1926). *J. industr. Hyg.*, 8, 466.
- Williams, Enid M. (1933). *The Health of Old and Retired Coalminers in South Wales*. Cardiff.

REPORTS: HER MAJESTY'S STATIONERY OFFICE, LONDON

- Registrar-General's Decennial Supplement, England and Wales, 1931. Part IIa, Occupational Mortality. London, 1938.
- Medical Research Council Special Report Series. No. 243 (1942), No. 244 (1943), No. 250 (1945).
- Ministry of Fuel and Power. Report of the Advisory Committee on the Treatment and Rehabilitation of Miners in the Wales Region Suffering from Pneumokoniosis (1944).
- Board of Trade. Provision of Employment in South Wales for Persons Suspended from the Mining Industry on account of Silicosis and Pneumoconiosis (1945). Cmd. 6719.
- Medical Research Council Memorandum No. 25. The Social Consequences of Pneumoconiosis among Coalminers in South Wales (1951). By P. Hugh-Jones and C. M. Fletcher.